Transmission of hepatitis B by a human bite: an occupational hazard

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Hepatitis B developed in a policeman 15 weeks after he was bitten on the hand. A few days after the bite hepatitis B developed in the assailant. The bite had drawn blood and this method of inoculation was presumed to be the route of transmission of the virus. Compensation was awarded on the grounds that this was an occupationally acquired disease.

Une hépatite de type B a été décelée chez un policier 15 semaines après qu'il eut été mordu à la main. Quelques jours après la morsure une hépatite de type B était aussi apparue chez son assaillant. La morsure avait fait couler le sang et on suppose que ce mode d'inoculation a servi de porte d'entrée au virus. Une indemnité a été accordée au policier en s'appuyant sur le fait qu'il s'agissait d'une maladie acquise dans l'exercice de ses fonctions.

Saliva is known to contain hepatitis B surface antigen (HB_sAg)[1,2] but its direct infectivity has not been demonstrated. A human bite that draws blood might be expected to be an effective route of transmission of hepatitis B by inoculation from the assailant to the victim but only one instance suggestive of this has been reported.[3] Since this mode of transmission should be recognized, and since it could represent an occupational hazard, we report a case in which the circumstantial evidence was strong that a human bite was the route of transmission of hepatitis B.

Case report

A 41-year-old policeman was admitted to another hospital Oct. 22, 1973. Ten days prior to this he had had a 1-day febrile illness followed by nausea and anorexia and then jaundice, dark urine and pruritus. During the previous year he had not received any injections, blood products or medications other than aspirin, and he was not aware of any contact with hepatitis. At that time the patient had forgotten that he had been bitten on the hand on June 20.

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He was jaundiced, had no fever and looked fairly well. The liver edge was palpable 2 cm below the right costal margin. Tests relevant to liver function yielded the following values: serum glutamic oxaloacetic transaminase (SGOT), 354 IU (normal, 0 to 35 IU); serum alkaline phosphatase, 240 IU (normal, 40 to 120 IU); and total serum bilirubin, 4.7 mg/dl (normal, < 1 mg/dl). During the next 2 weeks the total serum bilirubin value increased to 21.9 mg/dl and, although the SGOT had increased to 880 IU, a laparotomy was carried out on Nov. 7 because of the possibility of extrabiliary obstruction. Serum had been obtained Oct. 22 to test for HB_sAg. The result had not been reported at the time of laparotomy but subsequently was reported as positive.

Liver biopsy showed that the architecture was preserved, although strikingly distorted by enlargement of the portal tracts and swelling of the hepatocytes. The portal tracts contained proliferated bile ductules and a large number of inflammatory cells, mostly lymphocytes, with a few neutrophils and eosinophils. No piecemeal necrosis was seen but there was ballooning degeneration of the hepatocytes with single-cell necrosis. Many Councilman bodies were present. The central veins showed inflammation with centrilobular necrosis of hepatocytes. Cholestasis was mild and predominantly central. The features were those of acute icteric viral hepatitis.

His postoperative recovery was slow and on Nov 12 he was transferred to McMaster University Medical Centre for further management. On arrival he was unwell but improving. Total serum bilirubin value was 23 mg/dl; SGOT value was 144 IU. Serum obtained that day was positive for HB_sAg. He made an uneventful recovery. After 3 weeks his serum became negative for HB_sAg and the SGOT value returned to normal. Subsequent test results were normal. Tests for antimitochondrial antibodies, antinuclear factor and smooth muscle antibodies were negative and the patient remained in excellent health. There was no clinical evidence of hepatitis in his immediate family.

A claim was placed by the patient to the Workmen's Compensation Board of Ontario on the grounds that his illness had been acquired in the course of his occupation. Compensation was awarded.

Details of the bite and the assailant

While at McMaster University Medical Centre the patient remembered having been bitten on the hand June 20, 1973 by a man who was resisting arrest. The bite had broken the skin and drawn blood and the wound had been treated with a local

dressing. No other injury had occurred to either party. The assailant was believed at the time to be taking illicit drugs intravenously. Following his arrest he was committed to prison. Within 1 week the typical clinical picture of acute viral hepatitis developed and he was jaundiced on June 29. His SGOT value increased to 2280 IU and his total serum bilirubin value to 12 mg/dl. His serum was positive for HB_sAg; his saliva was not assayed for HB_sAg. He made an uneventful recovery and his serum became negative for HB_sAg.

Discussion

There was strong circumstantial evidence that the policeman contracted hepatitis by inoculation of virus from a human bite. His clinical illness started 16 weeks after the bite and he had only this brief contact with his assailant. The assailant was within a few days of the appearance of the clinical manifestations of hepatitis and his serum would have been positive for HB_sAg.[4] There was no other known contact with cases of hepatitis amongst the policeman's family or colleagues and no other recognized source of hepatitis. The possibility of transmission by inoculation was considered only in retrospect and epidemiologic studies that would have established this route more firmly were not undertaken. These include subtyping of the HB_sAg from both the policeman and his assailant, a prospective study of the policeman's serum for HB_sAg, and a study of his immediate family and contacts. However, the association between the bite and the development of hepatitis was thought to be sufficiently likely that an award for compensation was made on the grounds that this was an occupationally acquired disease.

MacQuarrie, Forghani and Wolochow[3] reported one somewhat similar case, in which a teacher at a school for mentally retarded children received puncture wounds on her finger from the teeth of one of her pupils while clearing his pharynx during an epileptic seizure. The pupil was known to be a chronic carrier of HB_sAg. Hepatitis developed in the teacher 5 months later and her serum became positive for HB_sAg of the same subtype. The degree of continuing contact between the boy and the teacher was not reported.

Both saliva and serum might be the fluid transmitting hepatitis B virus in a bite. HB_sAg has been demonstrated in the saliva of patients whose serum contains the antigen[1,2] but infectivity of saliva has not been demonstrated. Serum can infect by inoculation in amounts so small that it may be present in the mouth without overt evidence of injury.[5]

The history of the bite was obtained subsequent to laparotomy and liver biopsy; had the possibility of the bite as a method of transmission of hepatitis B been raised earlier, management might have been more conservative.

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